

CASE REPORT

Spontaneous internal carotid dissection in a 38-year-old woman: a case report

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This case report describes a patient found to have amaurosis fugax as a result of non-traumatic internal carotid dissection. Monocular blindness can be due to multiple causes including keratitis, acute glaucoma, vitreous hemorrhage, uveitis, retinal vascular occlusion, retinal detachment, optic neuropathy, trauma, or vascular malformations. In the setting of headache, neck pain, and an otherwise normal ophthalmic examination, this case report highlights the importance of recognizing transient ischemic attack and carotid artery dissection in the differential diagnosis. To further clarify the diagnosis, carotid ultrasound may aid diagnosis as was seen in this case, where decreased internal carotid artery velocities were found and subsequent CT angiography of the neck confirmed a diagnosis of carotid dissection. If a dissection is present, progression of symptoms may indicate impending cerebral infarction and warrant immediate attention. Antiplatelet therapy is the first-line treatment with anticoagulation, thrombolysis, and surgery reserved for cases of recurrent, progressive symptomatic episodes. Surgical options include endovascular repair such as angioplasty, stent placement, embolization, surgical revascularization, and bypass.

Keywords: *spontaneous carotid dissection; amaurosis fugax; ICA dissection; monocular blindness; transient ischemic attack; non-traumatic internal carotid artery dissection*

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Carotid dissection may be traumatic or spontaneous, resulting in hemorrhage within the arterial wall. Atraumatic causes are spontaneous, resulting in the absence of an external force (rapid or prolonged head movement) such as with exertion of the neck. Atraumatic dissections appear different than those of trauma, with the former involving the common carotid, resulting in complete occlusion and less likely to resolve. Systemic hypertension and arteriopathy, such as fibromuscular dysplasia, Marfan's syndrome, type 4 Ehlers-Danlos syndrome, and cystic medial necrosis, atheroma, and pheochromocytoma are common causes of spontaneous carotid dissection (1).

In the majority of patients with carotid dissection, symptoms include headache or neck pain. Internal carotid artery dissections have also been located more frequently near the carotid bifurcation, more commonly at the supraclinoid site and cervical segments of C1–C2 (1). In cases where primary carotid dissections are noted, careful examination for other regions is imperative, as 20% of cases will have multiple dissections. As previously noted, hemorrhage within the carotid wall is more commonly

a result of intimal injury, although hemorrhage from the vasa vasorum into the media is possible.

Formations of a hematoma dissecting through the media result in narrowing of the lumen or formation of a pseudoaneurysm. CT angiography has been the gold standard for diagnosing carotid dissection when the cervicocephalic region is involved, with magnetic resonance angiogram (MRA) gaining favor. Imaging may reveal dissection of a pseudoaneurysm or an intimal flap from elevation of the intima from extravasated blood, as described in this case. Irregular or smooth, tapered narrowing of the lumen may be seen, making it difficult to diagnose on initial imaging (1). In cases where vertebral artery involvement and subarachnoid hemorrhage are suspected, CT angiogram should not be replaced with MRA as it is more sensitive to this injury. Sonography is less reliable than CT angiogram and MRA; however, it is patient friendly and less expensive. Findings on sonography may consist of echogenic intimal flap and echogenic thrombus, with dissected region located distal to the visualized region on sonogram. Abnormal sonographic findings may also include decreased velocity of the

carotid bulb with the presence of high resistance of a bi-phasic pattern. Carotid dissections typically resolve in more than 80% of cases, with stenosis resolving more than pseudoaneurysms. Recurrence is typically uncommon.

Management is usually non-surgical, with antiplatelets recommended for presentations of headaches, oculosympathetic paresis (Horner's syndrome), and bruits (2). In patients with recurrent or progressive transient ischemic symptoms or strokes, despite being on aspirin, anticoagulation, or thrombolytics, surgical intervention should be considered.

Case report

A 38-year-old woman with a history of tobacco abuse and oral contraceptive use presented to the emergency department with transient loss of vision in the right eye, which lasted for 30 min, with a second episode that respected her horizontal meridian. The patient initially developed a non-productive cough for 2 weeks, followed by right-sided neck pain prior to the episode of vision loss. Vital signs were notable for a blood pressure of 124/85, heart rate 63 bpm, and respiratory rate 16. Eye examination revealed vision 20/20, 3 mm pupils, equal and brisk to direct and consensual light, reactive to accommodation. Cardiopulmonary examination revealed regular rate and rhythm, with a 2/6 systolic murmur loudest at the left lower sternal border, without rubs or gallops, and lungs were clear to auscultation. Neurologic examination revealed intact cranial nerves, no loss of hemifacial sweating, and preserved strength in all extremities. Electrocardiogram revealed normal sinus rhythm, no acute ST changes. Computed tomography of the head was negative. The patient was discharged home with a follow-up appointment with an ophthalmologist, who noted a normal eye exam with an unremarkable external and dilated retinal exam. The patient was referred to cardiology for cardiovascular workup. Carotid ultrasound revealed decreased velocity in the



Fig. 1. Dissection of the right internal carotid artery, which was the etiology for amaurosis fugax. This is noted as a string sign, representing the extent of the dissection.

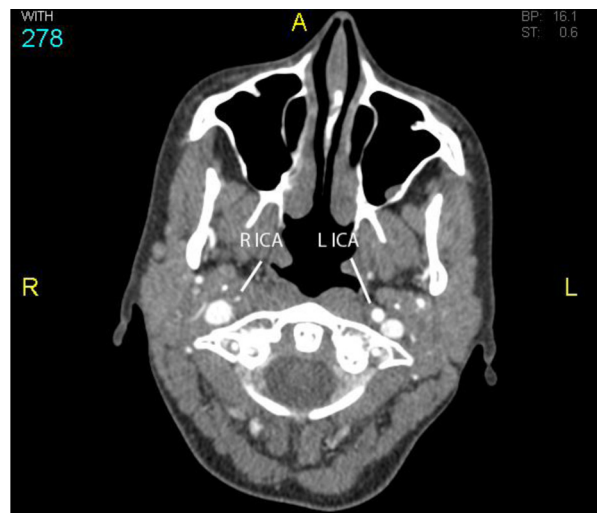


Fig. 2. Dissection of the right internal carotid artery is noted in a transverse view.

right internal carotid artery with evidence of an intimal flap. CT angiogram of the neck confirmed diffuse narrowing and string sign of the right intracranial internal carotid artery due to carotid dissection (Figs. 1 and 2). The right ophthalmic artery was not well visualized. The patient was started on aspirin, and she tolerated it well.

Discussion

This case describes the initial signs and symptoms of carotid dissection, with resultant amaurosis fugax. Notably, this patient did not have a history of diabetes, hyperlipidemia, hypertension, or migraines which further warranted workup for an atypical source of symptoms. The majority will have symptoms of neck pain and headache (>90%), followed by focal hemispheric ischemic symptoms (50–90%), Horner's syndrome (<50%), monocular blindness (6–30%), and dysgeusia (10–19%) (3). The incidence of carotid dissection is estimated to be two to three cases per 100,000 for all age groups, with a mean age of onset noted to be early 40s (4). Of those cases, amaurosis fugax is seen in 12%. There are four types of amaurosis fugax: embolic, hypoperfusion, angiospasm, and idiopathic (5).

Given the most common symptoms being non-specific, early diagnosis can be difficult, as symptoms are overlooked. Common non-traumatic causes of carotid dissection include fibromuscular dysplasia, Marfan's syndrome, atheroma, cystic medial necrosis, and systemic hypertension. In cases with carotid dissection, hemorrhage into the carotid wall could lead to intimal injury. Hematomas may form as the dissection progresses into the media, which can occlude the lumen. Early diagnosis and treatment can decrease the progression of the dissection. Diagnosis is typically initiated with carotid ultrasound as symptoms raise suspicion for source of possible emboli. Internal

carotid artery dissection can produce cerebral infarction in 36–68% of patients, as occlusion of the lumen near the dissection or embolization of thrombus occurs (3). An echogenic intimal flap or thrombus, along with decreased velocity of the carotid artery, may be noted on Doppler ultrasound. CT angiogram is the gold standard for diagnosing cervico-cephalic carotid dissection, noting tapered narrowing of the lumen.

In reference to the American Heart Association, initial treatment of aspirin 75–325 mg daily is recommended over anticoagulation with coumadin. Clopidogrel and aspirin with extended release dipyridamole may be considered in patients with aspirin allergy. Anticoagulation may be considered in patients with recurrence of symptoms, especially in the setting of a mechanical valve or atrial fibrillation. Treatment regimen also includes medication for lowering of blood pressure to less than 140/90 and LDL to less than 100, medication for diabetes mellitus, along with smoking cessation (6). In cases of recurrence despite appropriate anticoagulation with coumadin, surgical intervention may be considered. Surgical options include endovascular repair such as angioplasty, stent placement, embolization, surgical revascularization, and bypass.

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